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The effect of mating system on invasiveness: some genetic load may be advantageous when invading new environments

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Abstract The role of adaptation in determining invasion success has been acknowledged recently, notably through the accumulation of case studies of rapid evolution during bioinvasions. Despite this growing body of empirical evidence, there is still a need to develop the theoretical background of invasions with adaptation. Specifically, the impact of mating system on the dynamics of adaptation during invasion of a new environment remains only partially understood. Here, we analyze a simulation demo-genetic model of bioinvasion accounting for partial asexuality rates. We simulate two levels of recurrent immigration from a source population at mutation–drift–selection equilibrium to a new empty environment with a different adaptive landscape (black-hole sink). Adaptation relies on a quantitative trait coded explicitly by 10 loci under mutation, selection and genetic drift. Using this model, we confirm previous results on the positive effects on invasiveness of migration, mutation and similarity of local phenotypic optima. We further show how the invasion dynamics of the introduced population is affected by the rate of asexuality. Purely asexual species have lower invasion success in terms of probability and time to invasion than species with other mating systems. Among species with mixed mating systems, the greatest invasiveness is observed for species with high asexual rates. We suggest that this pattern is due to inflated genetic variance in the source population through the Hill-Robertson effect (i.e., clonal interference). An interesting consequence is that species with the highest genetic load in their source environment have greatest invasiveness in the new environment.

Keywords Mating system · Invasiveness · Niche evolution · Adaptation · Genetic load · Clonal interference · Source-sink dynamics · Hill-Robertson effect

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Introduction

Despite their negative impacts on biodiversity and economies, bioinvasions represent a great opportunity to study contemporary evolutionary processes (Sax et al. 2007). Native and introduced environments often

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differ in their ecological characteristics, so that adaptation is required for the introduced species to succeed (e.g., Sakai et al. 2001; Lee 2002; Muller-Scharer et al. 2004; Lambrinos 2004; Cox 2004). A fruitful source of theoretical results on this topic is the literature on adaptation to marginal habitats and the evolution of species ranges (e.g., Holt 1983; Holt and Gaines 1992; Kawecki 1995, 2000; Holt 1996; Holt and Gomulkiewicz 1997a, b; Kirkpatrick and Barton 1997; Gomulkiewicz et al. 1999; Holt et al. 2003; Travis et al. 2005; Bridle et al. 2010; Behrman and Kirkpatrick 2011; reviewed in e.g., Holt et al. 2005; Wiens and Graham 2005; Kawecki 2008). Notably, these studies have shown that adaptation to new habitats depends on complex interactions between dispersal, habitat quality, type and strength of selection, and genetic architecture of underlying traits.

Among the factors affecting the ability of a species to invade new environments is mating system (e.g., Brown and Burdon 1987). It has been stressed repeatedly that in species where sexual and clonal (parthenogenetic, apomictic) forms of reproduction coexist, clonal forms are often prevalent in marginal habitats—a phenomenon known as geographical parthenogenesis (reviewed in Kawecki 2008). For instance, the invasive plant *Rubus alceifolius* reproduces sexually in its native range (Southeast Asia) whereas it reproduces asexually in the recently invaded Madagascar and La Réunion islands (Amsellem et al. 2001). Similarly, emerging crop diseases caused by invasive fungi encompass numerous pandemics stemming from clonal populations, even in species with known mixed breeding systems in their native area. A classic example is the oomycete *Phytophthora infestans*, which has caused the historical great Irish famine in the mid-nineteenth century by devastating European potatoes (Desprez-Loustau et al. 2007). This has also been observed for other plant pathogens, e.g. rice blast caused by the ascomycete *Magnaporthe oryzae* (Saleh et al. 2012), and recently for the dramatic worldwide epidemic of wheat stem rust caused by race UG99 of the basidiomycete *Puccinia graminis tritici* (Singh et al. 2011). Asexuality is therefore considered as an important component of invasiveness (Hayes and Barry 2008).

There are a number of reasons why clonal populations may be successful invaders despite their lower genetic variability and greater genetic constraints. First, asexuality is often associated with a demographic advantage as a single individual is able to colonize an empty habitat, avoiding the risk of not finding mating partners (Pannell and Barrett 1998; Kolar and Lodge 2001; Pannell and Dorken 2006; Barrett et al. 2008). Second, in novel habitats, due to both randomness in the introduction process and the small population size, populations are thought to lack specialist parasites (enemy-release hypothesis, Keane and Crawley 2002; Liu and Stiling 2006), which, according to the Red Queen hypothesis, reduces the advantage of sex. Third, in some populations sexuality is thought to be advantageous because it ensures important ecological functions other than sex, e.g., winter survival or long-distance dispersal. Novel environments may be such that these functions are no longer a constraint, as suspected in the case of several emerging crop diseases (Saleh et al. 2012). Fourth, asexuality allows genotypes that happen to be locally adapted to be transmitted unaltered (Peck et al. 1998). So clonal individuals can be expected to be particularly invasive in situations where the adaptive challenge is small to non-existent. But asexuality also has shortcomings. Whatever the reason for their short-term success in marginal habitats, the lack of recombination makes asexual lineages prone to accumulate deleterious mutations that limit their evolutionary potential. This may compromise invasions that are associated with an adaptive challenge, i.e., when invaded habitats differ from native ones.

Most theoretical studies on the determinants of adaptability to a new environment have been generated in reference to purely asexual or sexual species or populations (reviewed in e.g., Holt et al. 2005; Kawecki 2008). Outside the context of bioinvasions, however, it has been shown that mixed mating systems can combine the advantages of both sexual and asexual reproduction in terms of genetic load reduction, fixation of beneficial mutations, and adaptation to fluctuating environments (e.g., Lynch and Gabriel 1983; Hedrick and Whittam 1989; Charlesworth et al. 1993; Green and Noakes 1995; reviewed in D'Souza and Michiels 2010). In addition, invading species from a diversity of phylogenetic groups have a mixed reproduction system. For instance this is the case in a great number of fungal plant pathogens (McDonald and Linde 2002; Desprez-Loustau et al. 2007). Many invasive plants also combine sexual reproduction with efficient vegetative reproduction systems (Barrett 2011). Last, in the invasive freshwater gastropod

Melanoides tuberculata, morphs that reproduce mainly asexually but with a small rate of sexuality display the greatest invasiveness (Facon et al. 2008).

Despite intuitions based on biological observations, we still lack profound insights into the role of partial asexuality on the success and dynamics of biological invasions. To investigate this topic, we performed a simulation study in which a population that is initially at mutation-drift-selection equilibrium in its native environment is allowed to colonize a new and different environment. Such a source-sink scenario has been used widely to study the causes of niche evolution and biological invasion (e.g., Gomulkiewicz et al. 1999; Kawecki 2000; Holt et al. 2003). This topic has been explored thoroughly in sexual species (monogamous hermaphrodites) by Holt et al. (2003), who confirmed that immigration plays a major role in adaptation to sink environments, and pointed out that immigration rates both too low and too high compromise the success of bioinvasions. They also showed that adaptation occurs via a 'punctuational' pattern where populations stay maladapted for a time and suddenly gain higher fitness in the new environment. Here, we elaborate on this model and specifically investigate the effect of the rate of asexuality on the probability and dynamics of invasions in new environments. We describe the specific assumptions made and the parameter space explored and then show that asexuality rate affects both the probability and speed of invasion in a non-monotonous manner. Species with a very low rate of sexual reproduction were found to display higher invasiveness than other species, all else being equal. Finally, we suggest that this effect is mediated mainly by the impacts of asexuality on the genetic diversity maintained in the source population.

Materials and methods

Model assumptions

We used an individual-based software called quantiNemo (Neuenschwander et al. 2008) that allows population dynamics to be simulated with concomitant evolution of quantitative traits with explicit genetic architecture. We modified this software to make it capable of simulating intermediate rates of sexual and asexual reproduction. The fixed and variable parameters used in the model are listed in Table 1.

Table 1 List of fixed and varying parameters of the model ('ind' stands for individual and 'gen' for generation)

Fixed parameters	Value
Number of loci (n)	10
Strength of selection ($1/\omega$)	1
Carrying capacity of source	1,000 individuals
Carrying capacity of sink	750 individuals
Burn-in phase*	1,000 generations
Simulation duration	500 generations
Fecundity (B)	5 ind. gen^{-1}
Varying parameters	**Values**
Environmental variance (σ_e^2)	{0, 1}
Mutation rate (μ)	{10^{-3}, 10^{-4}} locus^{-1}. gen^{-1}
Mutation effect variance (α^2)	{0.01, 0.05}
Distance between optimum (D)	{2.4, 2.8}
Migration rate (m)	{0.001, 0.005, 0.01} ind^{-1}. gen^{-1}
Asexuality rate (r)	[0, 1]

* The burn-in phase is the time lag to allow the source population to reach mutation–selection–drift equilibrium

We considered a closed source population of 1,000 diploid individuals, each characterized by a trait that confers local adaptation (Fig. 1). Recurrent migration occurs from this source environment to a new, initially empty, one. Both environments impose a stabilizing selection pressure on the trait (Gaussian fitness landscape) and differ in the optimal phenotype. Within each environment, the relative fitness w is thus given by:

$$w = \exp\left(-\frac{(z - z_{opt})^2}{2\omega^2}\right)$$

where z is an individual's phenotype, z_{opt} is the local optimal phenotype and ω determines the intensity of stabilizing selection. The distance between optima of the two environments is $D = |z_{opt2} - z_{opt1}|$. Without loss of generality, the source optimal phenotype (z_{opt1}) is fixed at 0 so that $D = |z_{opt2}|$. Following Holt et al. (2003), the trait is coded by $n = 10$ independent loci. The maximal number of alleles is limited to 255 per locus, with discrete allelic effects regularly spaced between -20α and 20α. The phenotype z of an individual is the sum of the 20 allelic values at the 10 diploid loci plus a random environmental component

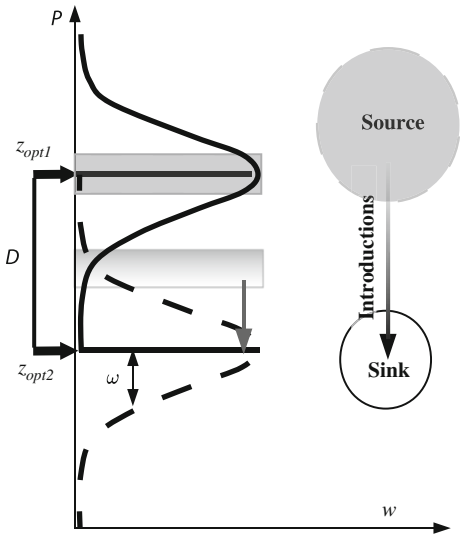

Fig. 1 Simulated scenario. A source population at mutation-selection-drift equilibrium sends migrants into a sink population. Sink and source populations have different optima. The immigrants (*grey-gradient rectangle*) might become adapted to their new environment (*grey arrow*) and consequently invade the sink population

(if present) that is sampled from a Gaussian distribution with variance σ_e^2. The life cycle consists of selection before migration and density regulation in both source and sink. We implemented the sequence of events as follows.

At each generation, the life cycle starts with reproduction. Within each environment, the software creates $BN\bar{w}$ offspring (rounded to the nearest whole number), where B is basic fecundity, N is the number of individuals in the preceding generation (parents), and $\bar{w}$ is the mean local fitness of the parents. There is no stochasticity at this stage. Then, each newly created individual is attributed a genotype by retrospectively sampling its parent(s) in the preceding generation. Selection occurs at this stage by considering that the probability of being sampled as a parent is $w_i/\sum_j w_j$ where w_i is the local fitness of individual i. Each offspring may be produced through asexual reproduction with probability r (i.e., asexuality rate). It is then genetically identical to its unique parent (provided no mutation occurs). Alternatively, offspring may be produced through sexual reproduction with probability $1 - r$. In the present simulations selfing is not allowed. The parents of a sexually produced individual are drawn without replacement from the local parental population. We checked on fewer replicate runs that the results were also valid with truly panmictic mating (results not shown). Once parents are chosen, offspring genotype is generated by choosing one allele at random within each parent, mimicking free recombination. The asexuality rate was varied among 9 values [0, 0.05, 0.2, 0.5, 0.8, 0.95, 0.999, 0.9999, 1]. Once offspring are produced, all the adults die.

After reproduction, mutations may occur within offspring genomes. The rate of mutation per individual per locus is μ. This parameter takes values 10^{-3} or 10^{-4}, which gives a genotypic mutation rate $2n\mu$ of 0.02 and 0.002, respectively. These settings are similar to those of Bürger and Lynch (1995) and Holt et al. (2003). We used the Increment Mutation Model (IMM) of quantiNEMO when a mutation occurs, a new allelic effect is drawn among the 255 possible allele states, following a discretized normal distribution of variance α^2 centered on the previous allele. When an allele out of the range of possible alleles is drawn ($<-20\alpha$ or $>20\alpha$), mutation does not occur. Given the high number of alleles, discretization is expected to cause little error. At mutation-selection-drift balance, under a Gaussian fitness landscape, the expected mutation effect on fitness results from the distribution of allelic mutation effects and the intensity of stabilizing selection: $E(s) = -\frac{\alpha^2}{2\omega^2}$ (Martin et al. 2006). Here, ω was arbitrarily fixed to 1. For the sake of comparison with Holt et al. (2003), we first set the variance of allelic mutation effects α^2 to 0.05 (which corresponds to an expected mutation effect on fitness of $E(s) = -0.025$) as in their study. Further, the expected mutation effect on fitness has been measured in *Saccharomyces cerevisiae* to be $\hat{s} \approx -0.01$ (Wloch et al. 2001). For our simulations to surround this value, we therefore explored a second value of $\alpha^2 = 0.01$ (which gives $E(s) = -0.005$).

At the migration step, each individual from the native population migrates with probability m into a new environment in which selection is also stabilizing with the same selection intensity but differs in optimal trait value, the distance between optima D being successively set to 2.4 and 2.8 as in Holt et al. (2003). We consider a black-hole setting to the extent that migration from the new environment back to the native one is not possible. The migration rate (m) varies among values 0.001, 0.005 and 0.01. Since

migration occurs before density regulation, the total number of individuals in the source that have a chance to migrate if all parents had maximal fitness (i.e. $\bar{w} = 1$) is $BN\bar{w} \approx 5,000$ because $B = 5$, $N = 1{,}000$. Thus, the maximal mean number of migrants per generation is equal to 5, 25 and 50, respectively, and the probability that no individual migrates at a given generation is at least 6.72×10^{-3}, 1.30×10^{-11} and 1.50×10^{-22}, respectively.

Finally, in both environments, local density regulation occurs if the local population exceeds carrying capacity (set at 1,000 and 750 in the native and invaded environments, respectively). Excess individuals are removed at random regardless of individual fitness.

Simulations

The source population initially comprised 1,000 individuals. To ensure rapid convergence of genetic variance to a mutation-selection-drift equilibrium, the initial population was built with maximal genetic diversity. Each allele in the initial population is chosen at random between 1 and 255 (reminding that allele effects are regularly spaced between -20α and 20α). As a consequence, the initial allele frequencies are approximately 1/255. During a burn-in phase of 1,000 generations, we let the source population reach a mutation-selection-drift equilibrium in the absence of migration to the sink. In a small preliminary assay, we checked that this burn-in period is sufficient for the source genetic variance to reach equilibrium, whatever mating system. For purely sexual populations, it was further shown to reach the expected mutation-selection-drift equilibrium (Bürger 1989; Fig. S2 Online Resource). After 1,000 generations, migration from the source to the sink begins, and we let this system evolve for 500 generations. For species with only one generation per year, this traces back to the European discovery of America. With 10 generations per year, as e.g., in fungal plant pathogens and other microbes, it brings us back 50 years, which seems reasonable for an invasion effect to be still detectable in population genetic data.

We have tested all combinations of the following variable and parameter values: asexuality rate, mutation rate, mutation effect variance, environmental variance, migration rate and distance between optima. For each combination of parameters, we ran 100 simulations. Prior to the simulations, the modified quantiNEMO algorithm was validated by repeating the simulations of Holt et al. (2003) (see Online Resource).

Measuring invasiveness

For a given combination of parameters, invasiveness has been characterized by estimating two parameters: invasion potential (*P750*) and time to invasion (*T750*). Invasion potential was estimated as the proportion of replicate runs for which the invasive population was at carrying capacity ($K = 750$ in the sink) at the end of simulations. Time to invasion is the time at which the invasive population has reached carrying capacity. By definition, it can be measured only on simulated populations that succeed in invading.

Describing population genetic variability

As a potential explanatory covariate of invasiveness, quantiNEMO was asked to compute the trait additive genetic variance, V_A, in the source population. Since purely additive effects are simulated (no dominance and epistatic effects), the additive genetic variance is identical to the total genetic variance. V_A results from the combined action of selection, drift, mutation rate, and mutation effects (Bürger and Lynch 1997). Because it determines the range of phenotypes available in the source population, it is likely to be an important factor of invasiveness. As a measure of linkage disequilibrium (LD), we used the statistic $|R|$ (Zaykin et al. 2008) that can be interpreted as the total correlation between a pair of loci. This statistic has been developed to provide an estimate as well as a test of LD in the multiallelic case. We averaged this statistic over all possible pairs of loci in order to get a genome-wide indicator of LD.

To understand how population genetic variability is affected by mutation, migration and mating system parameters, we tested which of the studied factors had the strongest explanatory power on the genetic variance in the source using type II Anova. The genetic variance was transformed by a Box-Cox transformation with parameter λ estimated by maximum likelihood. Interactions were not included in this model but we noticed that they have minor explanatory power compared to primary order effects (results not shown).

Results

Observed invasion dynamics

Typical simulations are shown in Fig. 2. It is worth noting that population increase during adaptation is very rapid but the time lag for this expansion to start is stochastic. Once exponential growth has started, the population quickly reaches its carrying capacity. Here over five replicate simulations, four exhibit successful invasion. In this example, *P750* is therefore equal to 4/5 and *T750* is less than 150 generations.

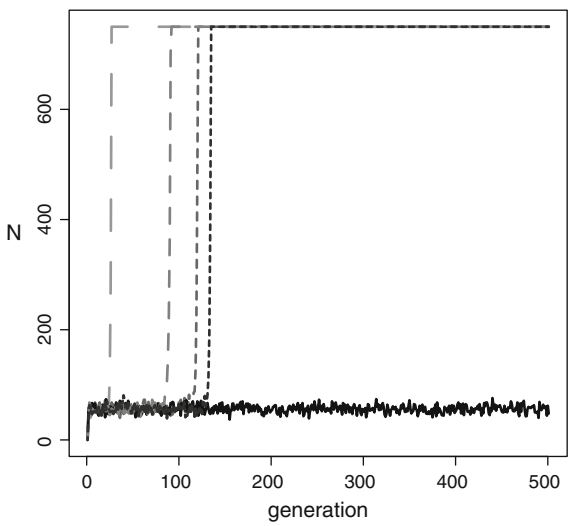

Fig. 2 Typical examples of population dynamics for populations adapting to a sink habitat. The population sizes shown are numbers of adults, after regulation and before reproduction. For the five examples shown, carrying capacities of the sink and source are 750 and 1,000, respectively. Other parameter values: $\sigma_e^2 = 0, \mu = 0.001, \alpha^2 = 0.05, D = 2.8, m = 0.01$ and $r = 0.5$. The mean number of migrants per generation is 50

Our simulations confirm previous studies of invasion in source-sink models (Fig. S3 Online Resource). As expected and as observed by Holt et al. (2003), the distance between optima is correlated negatively with invasiveness. We observe a positive relationship between environmental variance and invasiveness: environmental variance increases the chance of producing pre-adapted emigrants with a phenotype closer to the optimum of the sink population. Selection in the sink then acts on the settlers and will tend ultimately to genetically assimilate the adapted phenotype (Pigliucci and Murren 2003; Grether 2005; Kimbrell and Holt 2007). In the parameter range used here, we found higher migration rates to be associated with higher invasion potential. Finally, both higher mutation rate and variance of mutation effects improved invasion ability.

Effect of the rate of asexuality on invasiveness

In the present simulations, we observed that the proportion of successful invasions (invasion potential) was skewed towards higher values in completely sexual populations compared to completely asexual ones (Fig. 3) whereas the time to invasion had approximately the same distribution in both. On average, fully sexual populations are more invasive than fully asexual populations. However, the rate of asexuality was found to have a non-monotonous effect on invasion potential and time to invasion (Fig. 3).

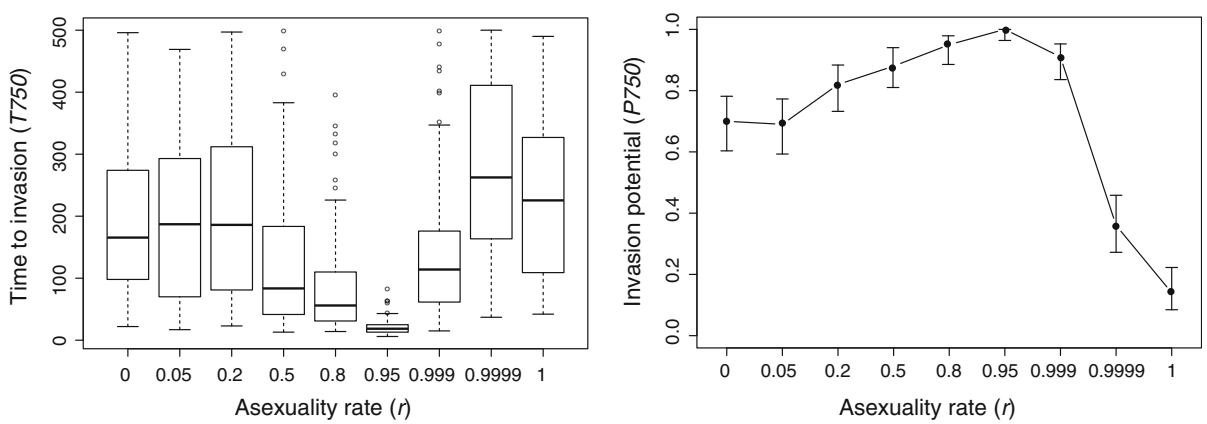

Fig. 3 Effect of asexuality rate (r) on the time to invasion (*T750*) and invasion potential (*P750*, i.e. the proportion of successful invasions). Parameter values: $\sigma_e^2 = 0, \mu = 0.001, \alpha^2 = 0.05, D = 2.8, m = 0.01$

The invasion potential increased with asexuality rate between 0 and 0.95. Above this range, invasion potential dropped dramatically and reached values close to zero for completely asexual populations. Invasions were also faster for higher asexuality rates up to a threshold of 0.95. Above this threshold, the time to complete invasion increased up to values close to 200 generations. To sum up, maximal invasiveness was reached neither for completely asexual nor for completely sexual populations but rather for a very high asexuality rate close to 1 ($\approx$0.95 in the present scenario and parameter range).

Invasiveness in relation to source genetic variability

Genetic variance is clearly correlated with the ability of a population to invade a new environment: populations with higher genetic variance had a higher invasion potential and shorter time to invasion (Fig. S4 Online Resource). Anova (which full results are shown in table S1 in Online Resource) revealed that model parameters explained 88.4 % of the variance of source genetic variance at the start of introductions. Mutation parameters (mutation rate and variance of mutation effects) had a positive and large influence on the genetic variance in the source (36.6 % of variance explained). Environmental variance also had a substantial, though smaller, positive effect (6.33 %). Logically, the distance between optima and migration rate had no effect on genetic variance in the source.

Asexuality rate had the greatest influence on source genetic variance (45.5 % of variance explained). To better understand these results, we plotted the genetic variance in the source as a function of asexuality rate (Fig. 4). Standing genetic variation is thought to be a key factor of rapid adaptation (Barrett and Schluter 2008), especially for invasive species (Prentis et al. 2008). We observed that the highest genetic variance was reached for rates of asexuality between 0.8 and 0.99.

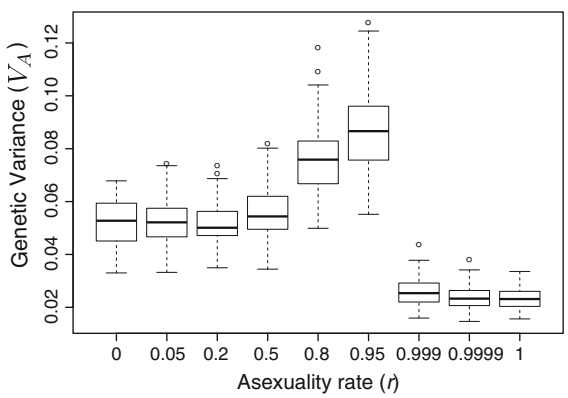

Fig. 4 Effect of asexuality rate (r) on genetic variance in the source population (V_A). Parameter values: $\sigma_e^2 = 0$, $\mu = 0.001$, $\alpha^2 = 0.05$

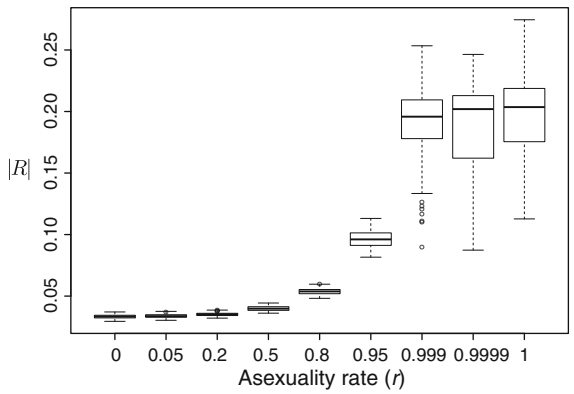

Fig. 5 Effect of asexuality rate (r) on linkage disequilibrium ($|R|$) in the source population. Parameter values: $\sigma_e^2 = 0$, $\mu = 0.001$, $\alpha^2 = 0.05$

Finally, the proxy for linkage disequilibrium $|R|$ was close to zero for an asexuality rate of between 0 and 0.5 (Fig. 5) and increases notably up to about 0.1 for an asexuality rate of 0.95. It reaches values close to 0.2 for very predominant asexuality (>0.95).

Discussion

Using individual-based stochastic simulations, we conducted a systematic exploration of the impact of asexuality rate on the probability of invasion and time to invasion. We have found that, using our simulation settings, purely sexual populations were better invaders than purely asexual ones. Moreover, there exists a non-monotonic relationship between asexuality rate and invasiveness, the best invaders being populations with highly predominant asexuality.

The genetic effects of mating system on invasiveness

In our attempt to better understand genetic issues, we have not considered the demographic advantages usually associated with either sexual (e.g., long-distance

dispersal, winter survival) or asexual (e.g., higher fecundity) reproduction. By their construction, our simulations give a slight demographic advantage to asexual reproduction over sexual reproduction because individuals are hermaphrodites but cannot self-fertilize. In other words, reproduction will fail if there is only one individual reproducing sexually in the population. Each generation, the probability of having only one migrant for migration rates 0.001, 0.005 and 0.01 is only 0.033, 3.28×10^{-10} and 7.57×10^{-21}, respectively. With more than one individual, reproduction failures due to the difficulty of finding a mate in low-density populations did not occur. Thus Allee effects are likely negligible in this study. A complete investigation into the impact of Allee effects on the adaptation to a sink environment is available in Holt et al. (2004) and Kanarek and Webb (2010). In the present study, fecundity was constant, whatever the proportion of offspring produced by asexual reproduction (no cost of males). Thus, only the genetic effects of the mating system were accounted for. There were thus two ways that mating system could affect invasion potential and time to invasion.

Firstly, mating system affects the genetic composition of the source population, hence the availability of genotypes that happen to be sufficiently fit in the sink environment to multiply once introduced. Sex breaks up linkage disequilibrium, making sexual populations capable of releasing additive genetic variance hidden by linkage disequilibrium (Otto and Lenormand 2002). Completely asexual populations cannot break linkage disequilibrium (Fig. 5), and rely only on mutations as a source of genetic variability. As a result, genetic variance at mutation-drift-selection equilibrium is lowest in such complete asexual populations (Fig. 4). In populations with high asexuality rate (i.e., close to 0.95), sex is too rare for linkage disequilibrium to disappear (Fig. 5). Thus, deleterious (locally maladapted) mutations can be associated with high-fitness mutations and the efficiency of selection against deleterious mutations and for advantageous mutations is reduced because of clonal interference (the so-called "Hill-Robertson effect", Hill and Robertson 1966; Felsenstein 1974). As a consequence, genetic variance is greater in such populations than in populations with even higher sexuality rates (Fig. 4). Populations with very predominant asexuality therefore have more mutations close to the sink environment optimum than do purely sexual species. The probability that migration samples a sink-adapted genotype is therefore higher and so is the probability of invasion.

Secondly, mating system may affect the evolutionary potential of the sink population. Again purely asexual species rely only on migration and mutation for locally adapted genotypes to appear, which limits their evolutionary potential (Burt 2000). Among species with a non-zero rate of sexuality, we observed that for a large range of asexuality rates ([0, 0.5]), invasive potential increases slightly while genetic variance in the source population remains quite constant (Figs. 3, 4). To further test this pattern, we made complementary simulations where the source population was always purely sexual, and asexuality rate varied in the sink population only. These simulations confirmed that invasion potential increases with increasing asexuality up to a rate of 80 % and then drops, while source genetic variance is constant (Fig. S6 Online Resource). This effect might be attributed to the potential of asexuality to protect good allele associations in the sink population. Under the introduction scenario considered here, migration constantly brings into the sink environment genotypes that are, on average, adapted to the source environment. In the sink environment recombination between local and migrant individuals may thus contribute to associate locally adapted mutations with mutation adapted to the source environment, slowing down adaptation to the sink optimum. This phenomenon is termed "migration load" and may contribute to maintaining local maladaptation in the sink (Kirkpatrick and Barton 1997; Travis et al. 2005). In this context, mixed reproduction has the advantage of being able to create new genotypes via recombination, which is more efficient than by mutation, and to protect good combinations from too extensive recombination with migrant genotypes.

Limits

We focused on invasions initiated by stochastic but recurrent migration from a source to a black-hole sink. Such invasions are thought to be more and more common as global trade increases with regular commercial routes that cause recurrent unidirectional migration events. A well-known example is the freshwater snail *Melanoides tuberculata* that has invaded Caribbean islands on multiple occasions,

likely through the transportation of ornamental aquarium plants (Facon et al. 2008). Invasions caused by recurrent migration are also becoming more frequent due to the ever-increasing human alteration of natural areas (Hufbauer et al. 2012). Human activities tend to open new habitats within native ones (e.g., crop fields, urban areas) making regular migration possible between the two habitats. Adaptation to human-altered areas may then pave the way for subsequent invasions at a global scale, as is thought to have occurred in various crop pathogens (Stukenbrock and McDonald 2008; Robert et al. 2012).

In the present study, we focused on a source population that is large enough not to be affected by genetic drift, at least for moderate asexuality rates. Consistently, the observed genetic loads in the source were low (Fig. S5 Online Resource). We made additional simulations varying source population size from 10 to 10,000 for mixed mating ($r = 0.95$) and purely sexual ($r = 0$) populations. The corresponding results are provided in figures S7 and S8 of the Online Resource, respectively. Both genetic variance and invasiveness decrease with decreasing population size under both mating systems. This effect is however much more pronounced for mixed mating populations, below 500 individuals.

It should be mentioned that in the present simulations we considered mating system to be a fixed characteristic of the source population; however, in many species mating systems may evolve. In particular, in species where both sexual and asexual modes of reproduction coexist, the level of investment into both types of reproduction may vary between individuals, and may thus be subject to selection. Plausible examples of such evolution at range margins exist (see Introduction). However, in most natural cases, it is hard to disentangle which evolutionary mechanisms have led to mating system modification (but see Roels and Kelly 2011 for a counter-example). For instance, in plant pathogenic fungi that have lost sex in invaded areas, we do not know at present whether such loss was adaptive—responding e.g., to the uniformity of agricultural landscapes—or caused by the random loss of one mating type during the bottleneck accompanying introduction (Saleh et al. 2012).

Theory meets comparative analyses

Mating system has been considered as a key determinant of invasiveness, with the ideal plant invader being seen as both self-compatible and capable of vegetative growth (Baker 1955). But the superiority of mixed mating systems over pure mating systems is questionable, notably because of the accumulation of non-conclusive comparative analyses. The ability to reproduce vegetatively (implicitly meaning a mixed mating system combining sexual reproduction and vegetative reproduction) has been shown to be associated with invasiveness in woody plants (Reichard and Hamilton 1997) and in plants from the Mediterranean islands (Lloret et al. 2005), but not in weeds (Sutherland 2004) and not in plants in general (Burns et al. 2011). The ability for clonal growth had no effect on lag phase duration in plant invasions (Larkin 2012). In a recent comparative analysis of invasiveness in fungal forest pathogens, Philibert et al. (2011) found that sexual reproduction (in general in addition to asexual reproduction) correlated with invasiveness (as previously suggested by McDonald and Linde 2002; Parker and Gilbert 2004). Thus it seems that, in fungal forest pathogens, mixed reproduction is a key trait for invasiveness.

Such conflicting results may be due partly to methodological issues. One is the difficulty of collecting information on mating systems from a sufficient number of invasive and non-invasive species to ensure sufficient power for statistical comparisons. A second difficulty is that, very often, the capacity for vegetative growth in plants or for sexual reproduction in plant pathogens correlates with other life-history traits. In weeds, Sutherland (2004) mentions that vegetative growth correlates strongly with life span, most vegetatively growing plants being perennial, which itself correlates with invasiveness. In fungal plant pathogens, sexual reproduction is often associated with winter survival or long distance dispersal. Beyond methodological aspects, these results also reveal that the effects of mating systems on invasiveness are diverse and context-dependent.

Here we show the superiority of very predominant asexual reproduction from a genetic point of view. If demographic features associated with mating systems were to be taken into account, it would probably affect the conclusion. But in any case, our model also suggests that, in future comparative analyses, it would be interesting to document rates of investment into asexual reproduction rather than simply the presence or absence of asexual reproduction.

A consequence for invasion biology

Up to now, conservation biologists have stressed that good invaders are populations that can rise to a double demographic and evolutionary challenge. Invasive populations are thought to have strong demographic features and/or the potential to adapt rapidly to their new environment (Prentis et al. 2008; Barrett et al. 2008). This is why authors have studied the importance of multiple introductions from populations with different environmental conditions (Dlugosch and Parker 2008) and hybridization with other species (Ellstrand and Schierenbeck 2000) as sources of variation in the invaded area.

Our study emphasizes that, in addition to its effect on the adaptive potential in the new environment, mating system affects the availability of genetic variability in the source environment: standing genetic variation is enhanced in the source of immigration by a mixed reproductive system with predominant asexuality. There is an interesting paradox here. In the situation that we considered here, where invasion is associated with an evolutionary challenge, the most invasive populations suffer from some genetic load (i.e., lower mean fitness) in their source environment (albeit moderate, Fig. S5 Online Resource). Because mating system acts as a constraint hampering local adaptation, these populations house a number of genotypes that are locally maladapted and happen to be better adapted to the sink environment. Therefore, the populations that have the greatest risks of becoming invasive elsewhere do not necessarily have superior demographic characteristics in their native environment. This result should, however, not be extended too far. Not all populations suffering from genetic load in their source environment are potential invaders. In particular, drift due to small population sizes may also hamper local adaptation, and generate genetic load. But it will also reduce the available genetic variance that critically determines invasiveness (Fig. S6).

Conclusion

From a conservation point of view, our study suggests focusing on species (plants, fungi and animals) where sexual reproduction occurs but with a high rate of asexual reproduction. It confirms that a mixed reproduction system gives the highest potential for rapid adaptation to a new environment. We have shown that the advantage of a mixed reproduction system may be associated with some genetic load in the native environment. Hence, contrary to what has sometimes been suggested (e.g., Williamson and Fitter 1996), species that are not particularly fit in their native environment compared to purely sexual or asexual species can still prove most aggressive invaders elsewhere. This result provides an additional hypothesis to explain the idiosyncrasy of biological invasions.

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